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U.S. EPA (U.S. Environmental Protection Agency). 2013. Methylmercury (MeHg) (CASRN 22967-92-6). Available: <http://www.epa.gov/iris/subst/0073.htm> [accessed 1 November 2013].

## ALSPAC Mercury Study and Fish Consumers

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Golding et al. (2013) described regression analysis of dietary contributions to maternal blood mercury levels, nested within the Avon Longitudinal Study of Parents and Children (ALSPAC). Fish intake explained only about 7% of the variance in blood mercury, leading them to conclude somewhat cautiously that “limiting seafood intake during pregnancy may have a limited impact on prenatal blood mercury levels” (Golding et al. 2013). The media, however, has been quick to over-interpret the results, and Golding herself was quoted:

We were pleasantly surprised to find that fish contributes such a small amount ... to blood mercury levels.... We hope many more women will now consider eating more fish during pregnancy. (ALSPAC 2013)

This is a much less cautious conclusion.

ALSPAC and Golding are responsible for many valuable publications on human development, but ALSPAC was not designed to investigate mercury exposure and effects. The categories of white fish, oily fish, and shellfish used by Golding et al. (2013) do not meaningfully reflect mercury content. With respect to the internal validity of the exposure estimate from the dietary questionnaire, high-mercury and low-mercury fish are represented among both the “white” and “oily” categories, and shellfish generally have very little mercury. Thus, internal validity is limited by a poor exposure metric.

External validity is more of a problem. The results of Golding et al. (2013) are not generalizable to the frequent consumers of fish who are most vulnerable to methylmercury exposure from fish during pregnancy. Women who ate fish frequently (> 3 times/week) made up < 2% of the ALSPAC subsample, and 60% and 76% consumed white fish or oily fish, respectively, no more than every 2 weeks (19% and 42% never consumed either). Moreover, < 1% had a blood mercury concentration > 5.8 µg/L, the level corresponding to the U.S. Environmental Protection Agency (EPA) methylmercury reference dose of 0.1 µg/kg/day (U.S. EPA 2013).

Although frequent fish consumers are a small part of the general population and the ALSPAC study, they are the population at risk for methylmercury exposure, particularly during pregnancy. Frequent fish consumers should reduce frequency or size of

fish meals or choose among the many types of fish low in mercury.

The mercury in fish is predominantly methylmercury, and for most people, fish is the only significant source of methylmercury (Björnberg et al. 2005; Mahaffey et al. 2004). Methylmercury is the toxic form that is almost 100% absorbed from the gut, and it is readily translocated to both the brain and the fetus. This is the reason for public health concern regarding consumption of high-mercury fish by frequent fish-eaters. At low levels of fish intake, other, non-organic forms and pathways influence blood mercury levels.

The results of Golding et al. (2013) suggest that women who rarely eat fish and already have a low blood mercury concentration will not further lower the mercury by eating even less fish. The study, however, did not address the risk for the frequent fish-eater who has elevated blood mercury.

It is not clear whether the developmental benefits ascribed to eating fish are due to its nutrients or to the healthy lifestyles that often correlate with eating fish frequently.

We conclude that women who eat fish rarely or never may benefit both self and fetus by eating fish occasionally. Pregnant women should choose low-mercury fish species, particularly if they eat fish frequently (more than twice a week).

*The authors declare they have no actual or potential competing financial interests.*

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## ALSPAC Mercury Study and Fish Consumers: Golding et al. Respond

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We apologize if our article (Golding et al. 2013) was unclear in some respects. We would like to respond to the comments of Gochfeld et al. and clarify some points in our article.

Contrary to the claims by Gochfeld et al., the Avon Longitudinal Study of Parents and Children (ALSPAC) study was designed to investigate the effects of various prenatal measures of the environment (including analyzing blood samples for trace metals and estimating dietary intake); the initial aim was to determine the relationship of these measures to a variety of different outcomes.

Gochfeld et al. state that “< 2% of the ALSPAC subsample” were women who ate fish frequently (> 3 portions a week). However, when consumers of both oily and white fish are combined, the frequent fish-eaters actually comprised 647 (18%) of our pregnant population.

In our article (Golding et al. 2013), we reported that “blood levels exceeded the 5.8 µg/L reference dose level ... in 38 women (0.92%).” A reanalysis of our data in regard to the 647 frequent fish consumers showed that only 2.0% of them had high mercury levels (> 5.8 µg/L).

In their letter, Gochfeld et al. cite two references (Björnberg et al. 2005; Mahaffey et al. 2004) to support their statement that “for most people, fish is the only significant source of methylmercury”—but neither of these studies assessed the contributions of mercury from any specific dietary source other than fish.

The categorical nature of our food frequency questionnaire successfully characterizes fish consumption in the study population; people do not eat the same type of fish regularly but typically consume different types of seafood. Internal validation of our fish exposure estimates shows that they are correlated with biological markers of fish consumption, such as omega-3 fatty acids (Williams et al. 2001). The measures we used reflected typical distributions of consumption of commercially available fish in the area. It should be noted that our results are based on population averages. Finer gradations in descriptions of fish consumption are not likely to substantially alter the results.

In response to the statement of Gochfeld et al. that our study “did not address the risk for the frequent fish-eater who has

elevated blood mercury,” we analyzed separately the group of frequent fish consumers (> 3 portions/week). We found that members of this group had a similar or lower contribution of blood mercury from seafood than the less-frequent consumers.

Gochfeld et al. suggest that published benefits of fish consumption may generally be a result of healthy lifestyles in general. In a previous study (Hibbeln et al. 2007), we performed a sensitivity analysis using paternal fish consumption. We found little association between offspring IQ (intelligence quotient) and paternal fish consumption compared with maternal fish consumption, implying that the maternal fish consumption effect was unlikely to be caused by social patterning.

As we note in our paper (Golding et al. 2013), detailed analyses of typical British diets have shown that fish provide between 25% (Ysart et al. 1999) and 33% (Ysart et al. 2000) of total dietary mercury. Thus, our figures are not as surprising as might have been expected.

We were surprised that Gochfeld et al. did not highlight the important finding of

contributions to mercury levels from foods that have no obvious nutrient value, such as herbal drinks, which we found to have an important association with blood mercury levels. Warning against such drinks may have greater net benefits to pregnant women than reduced fish consumption.

Finally, we agree with Gochfeld et al. that pregnant women might benefit slightly by substituting fish species containing very high methylmercury with those having lower levels, but we do not recommend that women cut down their overall fish consumption.

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